DISSERTATION ON

A STUDY ON ANALYSIS OF VARIOUS CAUSES OF HEMOPTYSIS AND THE SUCCESS RATE OF BRONCHIAL ARTERY EMBOLISATION IN THE FIRST SIX MONTHS FOLLOW UP PERIOD

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CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY ON ANALYSIS OF VARIOUS CAUSES OF HEMOPTYSIS AND THE SUCCESS RATE OF BRONCHIAL ARTERY EMBOLISATION IN THE FIRST SIX MONTHS FOLLOW UP PERIOD"** submitted by Dr. S. MURUGAN appearing for Part II M.D Branch I General Medicine Degree examination in April 2011 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamilnadu Dr. M. G. R. Medical University, Chennai. I forward this to the Tamilnadu Dr. M. G. R. Medical University, Chennai, Tamil Nadu, India.

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I, **Dr. S. MURUGAN** solemnly declare that this dissertation entitled **"A STUDY ON ANALYSIS OF VARIOUS CAUSES OF HEMOPTYSIS AND THE SUCCESS RATE OF BRONCHIAL ARTERY EMBOLISATION IN THE FIRST SIX MONTHS FOLLOW UP PERIOD"** is a bonafide work done by me at Madras Medical College and Government General Hospital from May 2010 to October 2010 under the guidance and supervision of my unit chief, **Prof. K.SIVASUBRAMANIAN, M. D.,** Professor of Medicine.

This dissertation is submitted to The Tamil Nadu Dr. M. G. R. Medical University, towards fulfillment of regulation for the award of M. D. degree (Branch- I) in General Medicine.

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CONTENTS

ANALYSIS OF VARIOUS CAUSES OF HEMOPTYSIS

INTRODUCTION

HEMOPTYSIS

Hemoptysis is defined as the expectoration of blood derived from the lungs or bronchial tree as a result of pulmonary or bronchial hemorrhage. [1]

Hemoptysis is a frightening symptom for patients and often is a manifestation of a significant underlying disease such as bronchogenic carcinoma. [2] Expectoration of even a relatively small amount of blood is an alarming symptom, and massive hemoptysis can be a life-threatening event. Therefore, hemoptysis of any degree needs thorough evaluation.

DIFFERENTIAL DIAGNOSIS OF HEMOPTYSIS

Source other than the lower respiratory tract

Upper airway bleeding

Gastrointestinal bleeding

Tracheobronchial source

Neoplasm

Bronchitis (acute or chronic)

Bronchiectasis

Broncholithiasis

Airway trauma

Foreign body

Pulmonary parenchymal source

Lung abscess

Pneumonia

Tuberculosis

Mycetoma

Good pasture syndrome

Idiopathic pulmonary hemosiderosis

Wegener’s granulomatosis

Lupus pneumonitis

Lung contusion

Pulmonary vascular source

Arteriovenous malformation

Pulmonary embolism

Elevated pulmonary venous pressure (esp. mitral stenosis)

Pulmonary artery rupture secondary to balloon-tip pulmonary artery catheter manipulation

Miscellaneous causes

Pulmonary endometriosis (catamenial hemoptysis)

Systemic coagulopathy or use of anticoagulants or thrombolytic agents

Medical reviews from different parts of the world revealed that the etiological pattern of hemoptysis has changed in the developed countries, with pulmonary tuberculosis becoming less important as a cause of bleeding from the lungs. In our country, however, the pattern has not changed. Pulmonary tuberculosis was the most common cause of hemoptysis four decades ago as shown by Rao in his study in 1960 and it is still the leading cause of it as is evident from the present study,

in which tuberculosis was found in 72% of patients with hemoptysis. Various studies from other developing countries have also shown pulmonary tuberculosis to be the major cause of hemoptysis. Studies from developed countries have shown malignancy and non-tuberculous causes to be the leading reasons for hemoptysis.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Hemoptysis is a nonspecific symptom and can occur in about 100 different clinical conditions. [3] In India, hemoptysis is almost synonymous with pulmonary tuberculosis and patients presenting with this symptom are often prescribed antitubercular treatment without proper workup. The purpose of this study was to identify the various etiologies of hemoptysis seen in our part of the country and show that not only active pulmonary tuberculosis but also old, healed pulmonary tuberculosis (sequelae) causes significant hemoptysis.

Differentiation between Hemoptysis and Hematemesis

	HEMOPTYSIS	**HEMATEMESIS**
HISTORY	Cough	Nausea/Vomiting
ASSOCIATION	Sputum	Food Particles
COLOUR	Bright Red	Dark Brown/Coffee Ground
FROTH	Present	Absent
REACTION	Alkaline	Acidic

TYPES OF HEMOPTYSIS

1. **Frank Hemoptysis**, in which the material coughed up consists of wholly of blood, occurs most commonly in bronchiectasis,

pulmonary infarction, tuberculosis, mitral stenosis, and also in bronchogenic carcinoma.

2. **Blood stained sputum**, in which the blood and sputum are intimately mixed up in various proportions, occurs most commonly in bronchogenic carcinoma.

3. **Blood streaked sputum**, in which streaks of blood are present in mucoid or purulent sputum, is a fairly frequent symptom in chronic bronchitis, but may also occur in bronchogenic carcinoma.

4. **Rusty sputum** in which degradation products of hemoglobin give the sputum a colour varying between rust and golden yellow, is a common feature of pneumococcal pneumonia and occurs in few other conditions.

BLOOD SUPPLY OF THE LUNGS

The bronchi, the connective tissue of the lung and the visceral pleura receive their blood supply from bronchial arteries, which are branches of the descending aorta. The bronchial veins (which communicate with the pulmonary veins) drain into the azygos and hemiazygos veins.

The alveoli receive deoxygenated blood from the branches of the pulmonary arteries. The oxygenated blood leaving the alveolar capillaries drains into the tributaries of the pulmonary veins, which follow the intersegmental connective tissue septa to the lung root. Two pulmonary veins leave each lung root and empty into the left atrium of the heart.

ETIOLOGY

The most common site of bleeding is the airway i.e the tracheobronchial tree, which can be affected by inflammation (acute or chronic bronchitis, bronchiectasis) or by neoplasm (bronchogenic carcinoma, endobronchial metastatic carcinoma or bronchial carcinoid tumor).

The bronchial arteries are the part of high pressure systemic circulation, are the source of bleeding in bronchitis, bronchiectasis or with endobronchial tumors.

Blood originating from the parenchyma can be either from a localized source such as infection (pneumonia, lung abscess and tuberculosis) or from a process diffusely affecting parenchyma (coagulopathy or Good pasture's syndrome).

Disorders primarily affecting the pulmonary vasculature include pulmonary embolic disease and those conditions associated with elevated pulmonary venous capillary pressure, such as mitral stenosis or left ventricular failure.

Although the relative frequency of different etiologies of hemoptysis varies from series to series, most recent studies indicate that pulmonary tuberculosis is still the commonest cause of hemoptysis in India.

Even after extensive evaluation, a sizeable proportion of patients have no identifiable etiology for their hemoptysis. These patients are classified as having idiopathic or cryptogenic hemoptysis and subtle

airway or parenchymal disease is presumably responsible for the bleeding.

PULMONARY TUBERCULOSIS

Also called adult type, reactivation, or secondary tuberculosis, post primary disease results from endogenous reactivation of latent infection and is usually localized to the apical and posterior segments of the upper lobes, where the substantially higher mean oxygen tension (compared with that in the lower zones) favours mycobacterial growth. In addition, the superior segments of the lower lobes are frequently involved. The extent of lung parenchymal involvement varies greatly, from small infiltrates to extensive cavitatory disease. With cavity formation, liquified necrotic contents are ultimately discharged into the airways, resulting in satellite lesions within the lungs that may inturn undergo cavitation. Massive involvement of pulmonary segments or lobes, with coalescence of lesions, produces tuberculous pneumonia. While up to one third of untreated patients reportedly succumb to severe pulmonary tuberculosis within a few weeks or months after onset (the classical galloping consumption of the past). Others undergo a process of spontaneous remission or proceed along a chronic, progressively debilitating course. Under these circumstances, some

pulmonary lesions become fibrotic and may later calcify, but cavities persist in other parts of the lungs. Individuals with such chronic disease continue to discharge tubercle bacilli into the environment. Most patients respond to treatment, with defervescence, decreasing cough, weight gain, and a general improvement in well being within several weeks.

Early in the course of disease, symptoms and signs are often non-specific and insidious, consisting mainly of fever and night sweats, weight loss, anorexia, general malaise, and weakness. However, in the majority of cases, cough eventually develops- often initially nonproductive and subsequently accompanied by the production of purulent sputum, sometimes with blood streaking. Massive hemoptysis may ensue as a consequence of the erosion of a blood vessel in the wall of a cavity. Hemoptysis, however, may also result from rupture of a dilated vessel in an old cavity (Rasmussen's aneurysm) or from aspergilloma formation in an old cavity.

Pleuritic chest pain sometimes develops in patients with subpleural parenchymal lesions. Extensive disease may produce dyspnoea and, in rare instances, adult respiratory distress syndrome. Physical findings are of limited use in pulmonary tuberculosis. Many

patients have no abnormalities detectable by chest examination, whereas others have detectable rales in the involved areas during inspiration, especially after coughing. Occasionally, rhonchi due to partial bronchial obstruction and classic amphoric breath sounds in areas with large cavities may be heard. Systemic features include fever (often low grade and intermittent) in up to 80% of cases and wasting. Absence of fever, however, does not exclude tuberculosis. In some cases, pallor and finger clubbing develop. The most common hematologic findings are mild anemia and leukocytosis. Hyponatremia due to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) has also been reported.

BRONCHITIS

Chronic Bronchitis is associated with chronic cough and phlegm. Risk factor being cigarette smoking, airway responsiveness, repeated respiratory tract infections, occupational exposure to coal mining, gold mining and cotton textile dust, air pollution and passive smoking. Persistent reduction in expiratory flow rates is the most typical finding in COPD. Patients with airflow obstruction related to COPD have a chronically reduced ratio of FEV1/FVC. Cigarette smoke exposure may affect the large airways, small airway(<2mm in diameter) and alveolar

space. Cigarette smoking often results in mucous gland enlargement and goblet cell hyperplasia. Patients with chronic bronchitis may have smooth muscle hyperplasia and bronchial hyper reactivity leading to airflow limitation. Three most common symptoms in COPD are cough, sputum production and exertional dyspnoea.

Patients with chronic bronchitis are quite frequently cigarette smokers and hemoptysis in these patients attributed to bronchitis without careful exclusion of neoplasm as a potential cause of bleeding. The hemoptysis in chronic bronchitis is most often blood streaking of sputum. Massive bleeding is unusual. In acute bronchitis, bleeding results from irritation of the unusually friable and vascular mucosa.

BRONCHIECTASIS

Bronchiectasis has been a common cause of hemoptysis. Bronchiectatic lungs show significant enlargement of the bronchial arteries as well as numerous and large anastamoses of the bronchial arteries with pulmonary arteries. The enlargement of the bronchial vessels is associated with the development granulation tissue and destructive rearrangement of the bronchial wall. Hemoptysis can be slight or massive and is often recurrent. Unusually associated with

purulent sputum or an increase in sputum purulence. Hemoptysis can, however, be the only symptom in so called dry bronchiectasis.

PNEUMONIA

Pneumonia is one of the causes of hemoptysis. The classic presentation of pneumonia is cough with fever with variable amount of sputum production, hemoptysis, dyspnoea and pleurisy.

In pneumococcal pneumonia the sputum is productive with blood streaked or rusty sputum. In pneumonia due to anaerobic bacteria, sputum is often purulent.

LUNG ABSCESS

Both acute and chronic lung abscess have a propensity for hemoptysis. After one to two weeks of infection, tissue necrosis with abscess formation or empyema occurs. Following cavitation, putrid sputum is noted in 50% or more of patients and hemoptysis may be seen.

In a large group of hemoptysis patients, lung abscess was responsible in approximately 5%. Bleeding may arise either from granulation tissues lining the abscess cavity or from a small branch of

pulmonary or bronchial arteries. A small frank hemoptysis may often precede the rupture of an abscess and the abscess contents are grossly blood stained during the first few days of rupture.

Certain radiologic signs described by Thomas are useful in identifying actual or impending hemoptysis.

1. Emptying and refilling of the abscess cavity on serial films.

2. Variations in the lucency and height of the air fluid level.

3. Variable parenchymal densities representing blood clots within the cavity.

In fungal infections also hemoptysis can occur. Pulmonary blastomycosis occurs in immuno compromised hosts. Within weeks to months, suppurative necrosis results in sputum production, which may be purulent and blood tinged. Endobronchial saprophytic pulmonary aspergillosis presents as chronic productive cough often with hemoptysis.

MITRAL STENOSIS

In mitral stenosis hemoptysis may result from a variety of causes. It is usually due to increased pulmonary venous pressure, pink frothy sputum may result from rupture of alveolar capillaries associated with acute pulmonary edema or from pulmonary infarction due to pulmonary embolism. Hemoptysis may be severe and profuse(pulmonary apoplexy). The edematous bronchial mucosa is more likely to be associated with chronic bronchitis resulting in blood stained sputum.

ANTICOAGULATION THERAPY

Long term anticoagulation therapy is associated with bleeding episodes. The incidence of minor bleeding is less than 2 to 3% per year. The incidence of major bleeding is 4 to 11% per year depending upon patient's age and associated comorbid conditions. The incidence of these complications is lower in those who receive low dose warfarin therapy(INR 2 to 3 vs >3).

The overall improvement in the anticoagulation control in the past 10 to 15 years has reduced the incidence of bleeding in these patients to about 0.2%. Review of observational and experimental studies showed annual bleeding rates of 0 to 4.8% for fatal bleeding and 2.4 to 8.1% for

major bleeding. Age is the main factor that increases the risk of bleeding.

Pulmonary hemorrhage is the most serious type of bleeding and varies from mild and occasional copious, recurrent, massive and fatal bleeding. Massive intrapulmonary hemorrhage is a common cause of death in Eissenmenger syndrome. Hemoptysis occurs most often in the presence of pulmonary vascular obstructive disease or in patients with extensive bronchial collateral circulation or pulmonary venous congestion.

PULMONARY THROMBO EMBOLISM

Dyspnoea is the most frequent symptom of pulmonary embolism and tachypnoea is its most frequent sign. Dyspnoea, syncope, hypotension or cyanosis indicates a massive pulmonary embolism. Pleuritic pain, cough or hemoptysis often suggests a small embolism located distally near the pleura.

Classic abnormalities in ECG include sinus tachycardia, new onset atrial fibrillation or flutter, and an S wave in lead I, a Q wave in lead III. Often the QRS axis is greater than 90. A normal or near normal chest Xray in a dyspnoeic patient suggests pulmonary embolism.

Abnormalities may be focal oligemia (Westermack's sign), a peripheral wedge shaped density above the diaphragm (Hampton's hump) or an enlarged right descending pulmonary artery (Palla sign). Computed tomography of the chest is the investigation of choice.

BRONCHIAL CARCINOID

Bronchial carcinoid is a rare tumor that resembles intestinal carcinoid tumor and is locally invasive eventually spreading to mediastinal lymph nodes and finally to distant organs. It is a highly vascular tumor that projects in to the lumen of a major bronchus causing recurrent hemoptysis.

BRONCHIAL ADENOMA

Bronchial adenoma are slowly growing, endobronchial lesions. Adenomas present in patients aged 15 to 60 years. Patients may have chronic cough, recurrent hemoptysis or obstruction with atelectasis.

BRONCHOGENIC CARCINOMA

Bronchogenic carcinoma is one of the common causes of hemoptysis especially in old age males. In squamous cell carcinoma hemoptysis may be minimal i.e., minimal streaking of blood in mucoid

sputum. Copious hemoptysis is uncommon. Squamous cell carcinomas are usually centrally located tumors. It usually occurs in smokers.

Adenocarcinomas are common in non smoking individuals and in female patients. Adenocarcinomas are usually peripherally located tumors, cough and hemoptysis are less common in peripheral neoplasms.

CHEST INJURIES

Hemoptysis follows a variety of chest injuries. Puncture of a lung by a fractured rib, contusion of a lung by severe blunt trauma to the chest and necrosis of the lining of the tracheo bronchial tree by inhaled fumes or smoke. Blunt trauma from the steering wheel during an automobile collision sometimes lacerates or fractures the tracheo bronchial tree. Stab or gunshot wounds often tear the lungs or airways resulting in hemoptysis.

BLEEDING DISORDERS

In hemophiliacs, pulmonary and pleural bleeding are uncommon, although mediastinal and pleural shadows have been noted radiographically and presumably originate from fresh or old hematomas. Patients with <1% factor 8 activity have severe disease. Patients with

levels 1 to 3% have moderate disease. Those with levels >8% have mild disease. Typically patients present with pain followed by swelling in a weight bearing joint, usually knee(hemarthrosis) leading to joint ankylosis. The most feared complications of hemophilia are oropharyngeal and central nervous system bleeding.

GOOD PASTURE'S SYNDROME

Good pasture syndrome is an auto immune disease in which auto antibodies are directed against type IV collagen. In 50% to 70% of patients have lung hemorrhage. Other features are hematuria, nephritic urinary sediment and sub nephrotic proteinuria.

WEGENER'S GRANULOMATOSIS

In wegener's granulomatosis, pulmonary involvement may be manifested as asymptomatic infiltrates or may be clinically expressed as cough, hemoptysis, dyspnea and chest discomfort. It is present in 85 to 90% of patients. In its classical form it presents as a characteristic triad of ELK i.e upper airway(ENT), lung(L) and renal disease(K-Kidney). It is characterised by necrotising granulomatous inflammation in the vessels in the ENT area and the lower respiratory tract.

SYSTEMIC LUPUS ERYTHEMATOSUS

In systemic lupus erythematosus most common manifestations is pleuritis with or without pleural effusion. Life threatening manifestations include interstitial inflammation leading to fibrosis and intraalveolar hemorrhage.

Lupus pneumonitis and diffuse infiltrative lung disease or interstitial lung disease can occur. Pulmonary hemorrhage is a rare but serious manifestation.

SNAKE BITE

Bleeding and clotting disturbances are seen after bites by vipers, pit vipers, Australian elapids and dangerously venomous colubrids. Snake venoms can cause hemostatic defects in a number of ways. Venom procoagulants can activate intravascular coagulation and produce consumption coagulopathy leading to incoagulable blood. For example, procoagulants in the venom of colubrids activate prothrombin and many crotalinae venoms have a direct thrombin like action on fibrinogen. Anticoagulant activity is attributable to venom phospholipases. Thrombocytopenia is a common accompaniment of systemic envenomations.

In patients bitten by Malayan pit vipers and green pit vipers there is initially inhibition of platelet aggregation followed by activation and the appearance of circulating clumps of platelets. Spontaneous systemic bleeding attributable to distinct venom components, hemorrhagins, which damage vascular endothelium. The combination of incoagulable blood, thrombocytopenia and vessel wall damage will result in massive bleeding, a common cause of death from viper bites.

BLEEDING MANIFESTATION IN CHRONIC KIDNEY DISEASE

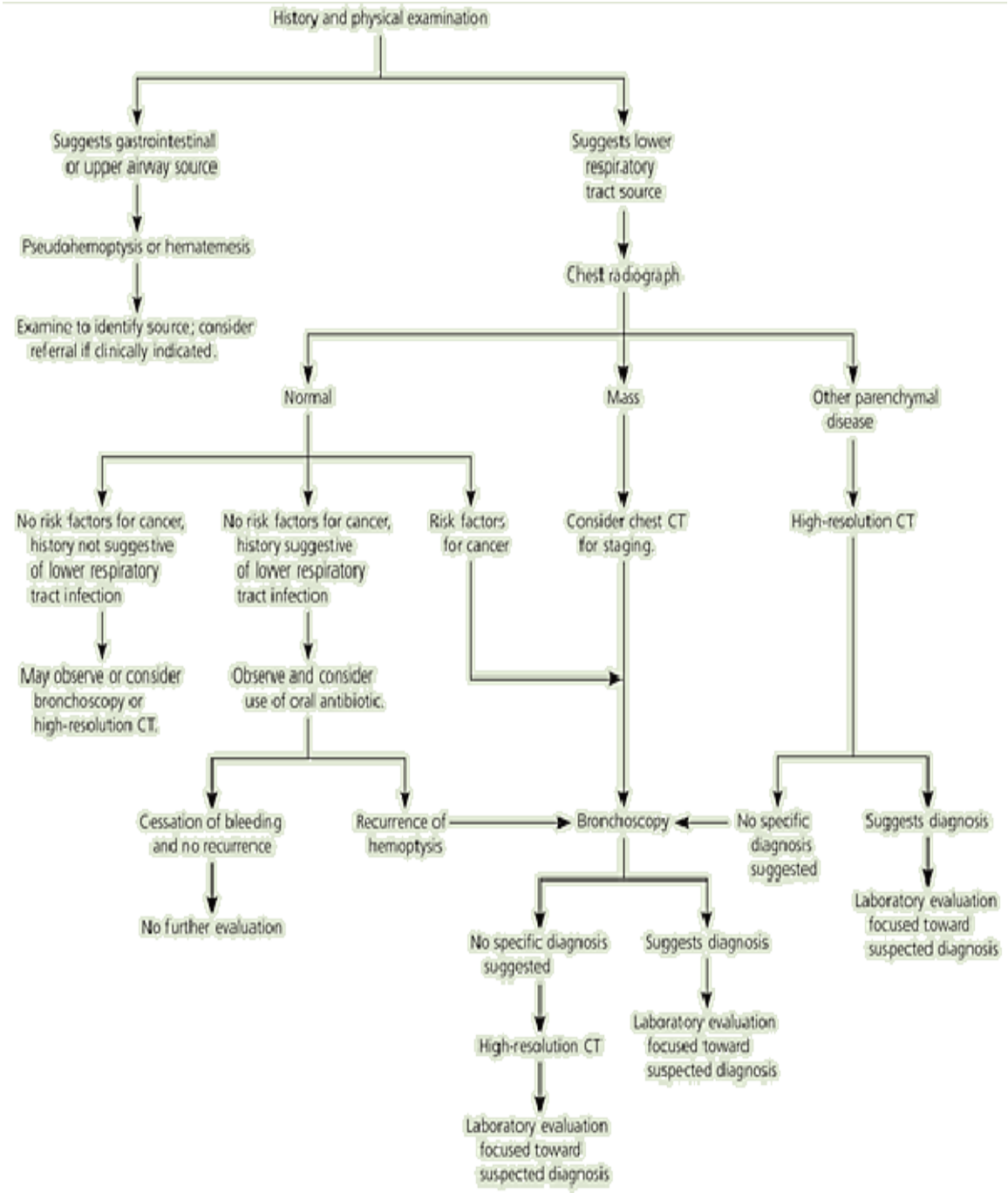
The etiology is multifactorial and that the defect mainly involves primary hemostasis. The impairement of primary hemostasis in uremia has two constant abnormalities, reduced adhesiveness of platelets and prolonged bleeding time. A direct correlation has been found between the prolonged bleeding time and clinical bleeding in uremic patients.

Antithrombin III is increased, while protein C anticoagulant activity is decreased, due to an inhibitor that interferes with anticoagulant properties of activated protein C. Though the platelet count is within the normal range, complex abnormalities in platelet functions have been noted. They include reduction in adenosine diphosphate and serotonin content, a defect in the mechanisms of

platelet secretion and release of alpha granule protein and beta thromboglobulin, increased platelet cyclic adenosine monophosphate and reduced generation of thromboxane A-2. Platelets from uremic patients have an impaired glycoprotein 2b-3a complex receptor function. Emerging evidence is also available that the bleeding tendency in uremia is related to an excessive formation of nitric oxide, an unstable, potent vasoactive molecule which also inhibits platelet function.

Hematological dysfunction may be an untoward side effect of drug treatment, and uremic patients are at an increased risk of bleeding of complications caused by drug therapy especially after administration of beta lactam antibiotics.

DIAGNOSING NON MASSIVE HEMOPTYSIS



DIAGNOSTIC CLUES IN HEMOPTYSIS:

PHYSICAL HISTORY	
Clinical clues	Suggested diagnosis*
Anticoagulant use Medication effect	Coagulation disorder
Association with menses	Catamenial hemoptysis
Dyspnea on exertion, fatigue, orthopnea, paroxysmal nocturnal dyspnea, frothy pink sputum	Congestive heart failure, mitral valve stenosis left ventricular dysfunction,
Fever, productive cough	Upper respiratory infection, acute sinusitis, acute bronchitis, pneumonia, lung abscess
History of breast, colon, or renal cancers	Endobronchial metastatic disease of lungs
History of chronic lung disease, recurrent lower respiratory track infection, cough with copious purulent sputum	Bronchiectasis, lung abscess
HIV, immunosuppression	Neoplasia, tuberculosis, Kaposi's sarcoma
Nausea, vomiting, melena, alcoholism, chronic use of nonsteroidal anti-inflammatory drugs	Gastritis, gastric or peptic ulcer, esophageal varices
Pleuritic chest pain, calf tenderness	Pulmonary embolism or infarction
Tobacco use	Acute bronchitis, chronic bronchitis, lung cancer, pneumonia.

Travel history	Tuberculosis, parasites (e.g., paragonimiasis, schistosomiasis, amebiasis, leptospirosis), biologic agents (e.g., plague, tularemia, T2 mycotoxin).
Weight loss	Emphysema, lung cancer, tuberculosis, bronchiectasis, lung abscess, HIV.

HIV = human immunodeficiency virus.

PHYSICAL EXAMINATION	
Clinical clues	Suggested diagnosis*
Cachexia, clubbing, voice hoarseness, Cushing's syndrome, hyperpigmentation, Horner's syndrome	Bronchogenic carcinoma, small cell lung cancer, other primary lung cancers
Clubbing	Primary lung cancer, bronchiectasis, lung abscess, severe chronic lung disease, secondary lung metastases
Dullness to percussion, fever, unilateral rales	Pneumonia
Facial tenderness, fever, mucopurulent nasal discharge, postnasal drainage	Acute upper respiratory infection, acute sinusitis
Fever, tachypnea, hypoxia, hypertrophied accessory respiratory muscles, barrel chest, intercostal retractions, pursed lip breathing, rhonchi, wheezing, tympani to percussion, distant heart sounds	Acute exacerbation of chronic bronchitis, primary lung cancer, pneumonia

Gingival thickening, mulberry gingivitis, saddle nose, nasal septum perforation	Wegener's granulomatosis
Heart murmur, pectus excavatum	Mitral valve stenosis
Lymph node enlargement, cachexia, violaceous tumors on skin	Kaposi's sarcoma secondary to human immunodeficiency virus infection
Orofacial and mucous membrane telangiectasia, epistaxis	Osler-Weber-Rendu disease
Tachycardia, tachypnea, hypoxia, jugulovenous distention, S3 gallop, decreased lung sounds, bilateral rales, dullness to percussion in lower lung fields	Congestive heart failure caused by left ventricular dysfunction or severe mitral valve stenosis
Tachypnea, tachycardia, dyspnea, fixed split S2, pleural friction rub, unilateral leg pain and edema	Pulmonary thromboembolic disease
Tympani to percussion over lung apices, cachexia	Tuberculosis

Laboratory Tests	Diagnostic findings
White blood cell count and differential count.	Elevated cell count and differential shifts may be present in upper and lower respiratory tract infections
Hemoglobin, hematocrit	Decreased in anemia
Platelet count	Decreased in thrombocytopenia
Prothrombin time, International Normalized Ratio, partial thromboplastin time	Increased in anticoagulant use, disorders of coagulation
Arterial blood gases	Hypoxia, hypercarbia
D-dimer	Elevated in pulmonary embolism
Sputum Gram stain, culture, acid-fast bacillus smear and culture	Pneumonia, lung abscess, tuberculosis, mycobacterial infections
Sputum cytology	Neoplasm
Purified protein derivative skin test	Positive - increases risk for tuberculosis
Human immunodeficiency virus test	Positive - increases risk for tuberculosis, Kaposi's sarcoma
Erythrocyte sedimentation rate	Elevated in infection, autoimmune disorders (e.g., Wegener's Granulomatosis, systemic lupus erythematosus, sarcoid, Goodpasture's syndrome), may be elevated in neoplasia

Chest radiograph finding	Suggested diagnosis*
Cardiomegaly, increased pulmonary vascular distribution	Chronic heart failure, mitral valve stenosis
Cavitary lesions	Lung abscess, tuberculosis, necrotizing carcinoma
Diffuse alveolar infiltrates	Chronic heart failure, pulmonary edema, aspiration, toxic injury
Hilar adenopathy or mass	Carcinoma, metastatic disease, infectious process, sarcoid
Hyperinflation	Chronic obstructive pulmonary disease
Lobar or segmental infiltrates	Pneumonia, thromboembolism, obstructing carcinoma
Mass lesion, nodules, granulomas	Carcinoma, metastatic disease, Wegener's granulomatosis, septic embolism, vasculitides
Normal or no change from baseline	Bronchitis, upper respiratory infection, sinusitis, pulmonary embolism
Patchy alveolar infiltrates (multiple bleeding sites)	Bleeding disorders, idiopathic pulmonary hemosiderosis, Goodpasture's syndrome

OBJECTIVE

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1. To analyse the various etiological factors of hemoptysis prevalent in our part of the country.

2. To show that not only pulmonary tuberculosis but also its sequelae is a major cause of hemoptysis .

MATERIALS AND METHODS

MATERIALS AND METHODS

Hundred consecutive patients of hemoptysis who were admitted to the medical wards of the Institute of Internal Medicine, Government General Hospital, Chennai were included in this study.

Type of study: Cross sectional study

Duration of study: May 2010 - October 2010

Statistical Analysis : SPSS Software

Inclusion criteria:

1. Inpatients of medical wards in GGH, Chennai- 3 with symptoms of hemoptysis
2. Age more than 14 years.
3. Both males and females are included in this study

Exclusion criteria:

1. Patients who have been embolised earlier and presenting with hemoptysis again.
2. Age less than 14 years

Approval from the institute's ethical committee and informed consent from all patients was obtained prior to commencement of the study. Demographic data, including sex and age, was collected. The amount of hemoptysis was also noted. Every patient was asked to collect the expectorated blood in a glass. The amount of hemoptysis was recorded and converted to a milliliter equivalent (i.e., one small glass = 100 ml). The episodes of hemoptysis were stratified into three groups according to the amount of blood expectorated, i.e., mild: < 100 ml/day; moderate: 100-300 ml/day; massive: >300 ml/day.

Patients were also grouped into categories on the basis of their primary diagnosis (like pulmonary tuberculosis, neoplasm, chronic bronchitis, bronchiectasis, others, etc). Diagnosis was made after thorough clinical evaluation and appropriate investigations like chest radiography, echocardiogram, sputum examination, computed tomography of thorax (CT), bronchoscopy, CT guided biopsy.

Diagnosis of pulmonary tuberculosis was based on chest radiography and sputum examination for acid fast bacilli. A chest radiograph demonstrating nodular, alveolar, or interstitial infiltrates predominantly affecting the upper zone of the lung(s) in

symptomatic patients (i.e., those with cough, weight loss, and fever with night sweat) were considered suggestive of active pulmonary tuberculosis. When the chest radiograph showed inactive processes such as calcified granuloma, and there were no symptoms other than hemoptysis, the patient was diagnosed as having inactive pulmonary tuberculosis. For bronchogenic carcinoma, diagnosis was based on histopathology. Bronchitis was diagnosed when a patient had symptoms consistent with upper airway infection and a normal chest radiograph. Diagnosis of bronchiectasis was confirmed by high-resolution CT of thorax.

Hemoptysis was categorized as

1. Mild (< 100 ml/day)
2. Moderate (100-300 ml/day)
3. Massive (>300 ml/day).

We also categorized the patients according to the primary etiology of the hemoptysis.

RESULTS

OBSERVATION AND RESULTS

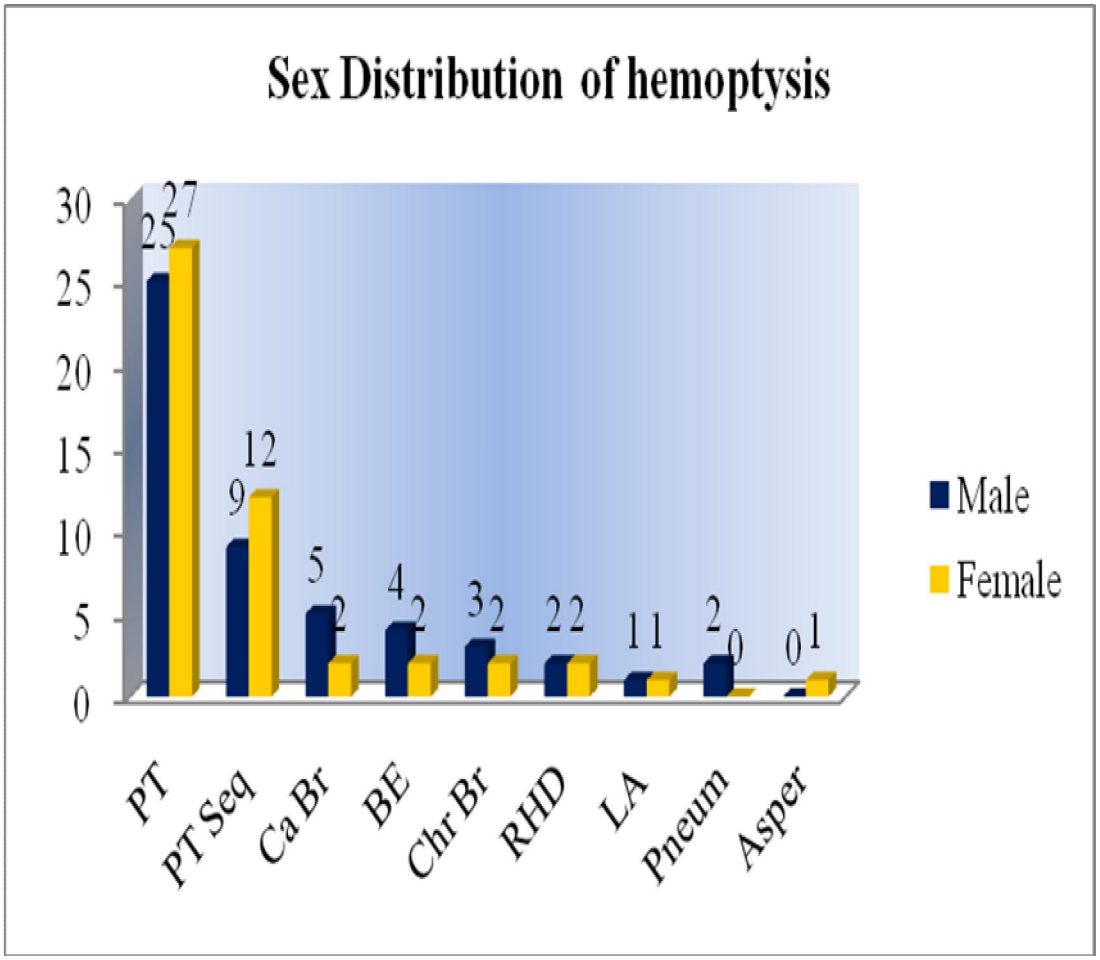
VARIOUS CAUSES OF HEMOPTYSIS

Diagnosis	No. of patients
Pulmonary tuberculosis	52
PT sequelae	21
Malignancy	7
Bronchiectasis	6
Chronic bronchitis	5
Rheumatic heart disease	4
Lung abscess	2
Pneumonia	2
Aspergilloma	1

Sex	No. of patients
Male	50
Female	50

Of the 100 patients with hemoptysis included in this study, 50 were males and 50 were females. Pulmonary tuberculosis was the

leading cause of hemoptysis. There were 73 patients in the pulmonary tuberculosis group (includes both active and inactive), 7 in the neoplasm group, 5 in the chronic bronchitis group, 6 in the bronchiectasis group, 4 were due to rheumatic heart disease, 2 were due to lung abscess and 2 were due to pneumonia and 1 due to aspergilloma.



From the above representation, we see that PT sequelae, chronic bronchitis and bronchiectasis seem to be more in males. All other conditions are almost equally distributed in both sexes.

Common causes of Hemoptysis

From the above bar diagram we see that active pulmonary tuberculosis is the major cause of hemoptysis contributing 50%, i.e., every second case of hemoptysis is due to PT. Also PT sequelae

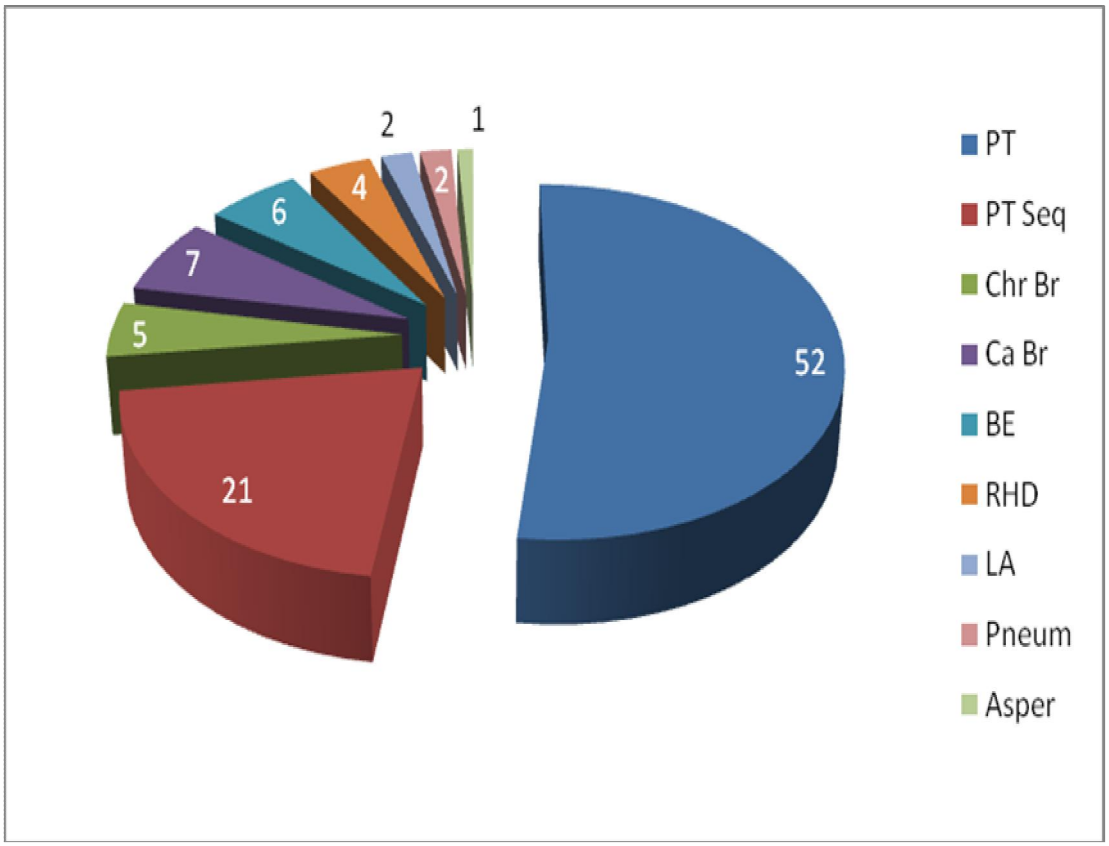
(inactive PT) ranks second in the list contributing about 21% of the cases. Other causes like bronchitis, malignancy which make up the huge chunk of cases in the western world are only a minor cause of hemoptyis in our country.

Statistical Analysis

	Observed N	Expected N	Residual
PT	52	11.1	40.9
PT Seq	21	11.1	9.9
Chr Br	5	11.1	-6.1
Ca Br	7	11.1	-4.1
BE	6	11.1	-5.1
RHD	4	11.1	-7.1
LA	2	11.1	-9.1
Pneum	2	11.1	-9.1
Asper	1	11.1	-10.1
Total	100		

p value is <0.001. It is significant at 1% level.

PIE CHART SHOWING CAUSES OF HEMOPTYSIS



CONCLUSION

CONCLUSION

1. Although pulmonary tuberculosis is the most important cause of hemoptysis in India, it may also occur due to a variety of other causes. Antitubercular treatment should not be started without proper diagnostic workup.

2. The fact that all who have hemoptysis do not have tuberculosis and all tuberculosis patients don't present hemoptysis should be kept in mind, always.

3. Another point of interest is that patients with hemoptysis need not always have active tuberculosis. About 20% of patients had hemoptysis due to PT sequelae/ inactive tuberculosis.

4. In our country, pulmonary tuberculosis and its sequelae, continues to be the predominant cause contributing to about 73% of cases of hemoptysis whereas malignancy and bronchitis seems to predominate the etiology list only in developed countries.

BRONCHIAL ARTERY EMBOLISATION

BRONCHIAL ARTERY EMBOLISATION

Life-threatening hemoptysis is one of the most challenging conditions encountered in critical care and requires a thorough and timely investigation. Despite advances in medical and intensive care unit management, massive hemoptysis remains a serious threat. According to recently published data, 28% of chest clinicians had experienced a patient's death from massive hemoptysis during a previous 1-year period (6). Conservative management of massive hemoptysis carries a mortality rate of 50%–100% (7). The cause of death is usually asphyxiation, not exsanguination (8). The reported mortality rates for surgery performed for massive hemoptysis range from 7.1% to 18.2% (9). However, the mortality rate increases significantly, up to about 40%, when the surgery is undertaken as an emergency procedure (10). Bronchial artery embolization (BAE) has become an established procedure in the management of massive and recurrent hemoptysis; its use was first reported in 1973 by Remy et al (11). The efficacy, safety, and utility of BAE in controlling massive hemoptysis have been well documented in many subsequent reports (11-19). Because of poor pulmonary reserve and other medical comorbid conditions, most patients with massive hemoptysis are not surgical candidates (7,8).

However, surgery remains the procedure of choice in the treatment of massive hemoptysis caused by specific conditions, such as hydatid cyst, thoracic vascular injury, bronchial adenoma, and aspergilloma that is resistant to other therapies (20). Even in surgical candidates, BAE is effective in preparing the patient for elective rather than high-risk emergency surgery (9).

AIM OF THE STUDY

AIM OF THE STUDY

To determine the success rate of Bronchial Artery Embolisation, performed for patients of massive hemoptysis (due to pulmonary tuberculosis or its sequalae), during the first six months follow up period.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The source of massive hemoptysis is usually the bronchial circulation (90% of cases) rather than the pulmonary circulation (5%) (23). In a minority of cases (5%), massive hemoptysis may originate within the aorta (eg, aortobronchial fistula, ruptured aortic aneurysm) or the systemic arterial supply to the lungs (24–26). In many acute and chronic lung diseases, pulmonary circulation is reduced or occluded at the level of the pulmonary arterioles because of hypoxic vasoconstriction, intravascular thrombosis, and vasculitis (27). As a result, bronchial arteries proliferate and enlarge to replace the pulmonary circulation. The enlarged bronchial vessels, which exist in an area of active or chronic inflammation, may be ruptured due to erosion by a bacterial agent or due to elevated regional blood pressure. The arterial blood under systemic arterial pressure subsequently extravasates into the respiratory tree, resulting in massive hemoptysis (27,28). Bronchial artery embolization is mainly performed for control of life-threatening massive haemoptysis (expectoration of blood). It can also be performed for patients with recurrent episodes of small volume hemoptysis, who are not fit for surgery, although 20% of these patients will rebleed within next 6 months.

The long-term success rate is 45% to 58%. Immediate control of haemorrhage is achieved in 75% to 95% of patients.

MATERIALS AND METHODS

MATERIALS AND METHODS

Type of study: Follow up study

Duration of study: May 2010 to October 2010

Statistical Analysis : SPSS Software

Inclusion criteria

1. All tuberculosis patients who have been admitted in the medical wards of GGH, Chennai- 3 for complaints of hemoptysis.
2. Both active and inactive tuberculosis patients are included in this study.
3. Hemodynamically stable patients.
4. Aged above 14years.

Exclusion criteria

1. Hemoptysis not of TB origin.
2. Patients who have been embolised earlier for similar complaints.

Twenty patients between the ages of 16 and 65 years, with a mean age of 40.5 years formed the group. There were 15 men and 05 women. All the patients had massive hemoptysis and were been treated for tuberculosis in the past/present. The criteria for massive hemoptysis were:

More than 300 ml of blood in 24 hours.

More than 100 ml per day for more than 2 days.

Procedure

• Before the procedure, chest X-ray, computed tomography (CT) and bronchoscopy was performed to locate the bleeding site.

• Under X-ray fluoroscopic guidance, angiography with digital subtraction facility was performed for delineation of vascular structures. The arterial access was usually via the common femoral artery at the groin region. Approach via brachial artery or radial artery at the upper limb was occasionally needed.

• The bronchial artery and the other arteries supplying the bleeding sites was identified and selectively cannulated with catheter. A smaller co-axial catheter through the original catheter was commonly used for

superselective catheterization. The spinal artery was avoided or bypassed if it was identified.

• Particles were then injected through the catheter to block the arteries. Gel foam was used.

• The procedure usually required 2-4 hours.

• After the procedure, vital signs (e.g., blood pressure and pulse rate) were monitored Cavitatory and non-cavitatory lesions were seen in 12 and 8 patients respectively. Patients were followed up for two days as in-patients and thereafter for six months as outpatients.

Potential Complications

With the use of non-ionic contrast medium, coaxial catheters and digital subtraction angiographic technique, serious complications arising from bronchial artery embolization is not common.

• Particles flowing to the spinal artery, causing spinal artery occlusion and resulting in paralysis of the legs and lower part of the body – very rare.

• Injury of bronchial artery with life-threatening bleeding – rare.

• Non-target embolization of branches of subclavian artery causing injury to other organs: brainstem, fingers – rare.

• Chest pain and difficulty in swallowing: These may occur 2-7 days after embolization and are usually self-limiting.

• Vascular damage due to arterial puncture or manipulation of guidewire and catheter: rare.

• Transverse myelitis (inflammation of the spinal cord): rare.

• Fistula formation between the bronchus and esophagus: rare.

• Death of bronchial tissue: rare.

• Procedure related death is rare.

• The overall adverse reactions related to iodine-base contrast medium is below 0.7%.

The mortality due to reaction to non-ionic contrast medium is below 1 in 250000.

RESULTS

OBSERVATION AND RESULTS

Control of bleeding	No of patients
Immediate control	14
Recurrence within 24 hrs	1
Recurrence > 24 hours	4

Therapeutic embolization was achieved in 19 patients. In one, the required artery could not be catheterized. Dilated, tortuous bronchial arteries were seen at the site of lesion. Patients with bronchial to pulmonary artery shunting were also embolised. The average time taken for the procedure was forty-five minutes.

In one patient, spinal arteries were found to originate from a major bronchial artery. In this case, the catheter was negotiated beyond the origin of the spinal arteries.

The criterion for successful embolization was expectoration of less than 100 ml of blood in the 24 hours following the procedure.

Occasional blood stained sputum was seen up to one week following the procedure and cessation of this was considered success.

Hemoptysis of more than 100ml in twenty-four hours was considered as treatment failure.

These patients were followed up for a period of six months. They were instructed well to contact us in case of any recurrence of symptoms and to get hospitalized at the earliest. Of the 19 patients who underwent BAE procedure, 14 of them stayed asymptomatic throughout the six month follow up period, 1 developed hemoptysis within 24 hours of the procedure and 4 experienced hemoptysis during the six month follow up period.

Post procedural hemoptysis

	Observed N	Expected N	Residual
Present	5	9.5	-4.5
Absent	14	9.5	4.5
Total	19		

p value is 0.039. It is significant at 5% level.

DISCUSSION

Most of the patients with massive hemoptysis are poor surgical candidates as they lack respiratory reserve [32]. Bronchial artery embolization provides effective management in patients, when massive hemoptysis results from the sequelae of tuberculosis rather than complications of the disease [33],[34]. The bronchial arteries are the most common source of massive hemoptysis in these patients. This is usually due to chronic bronchial inflammation leading to bronchial artery hypertrophy and an aneurysmal dilatation of these vessels [35], [36].

On angiography, the major source of hemorrhage is the bronchial vessel and hypervascularity is the single most common pathological abnormality on the affected side [37],[38]. Generally, cavities are the source of hemoptysis and the arteries supplying these cavities were always embolised [39]. The presence of the anterior spinal artery as a branch of the costocervical trunk was not considered to be a contraindication for the procedure [40].

According to our study, BAE success rate is 74%.

CONCLUSION

CONCLUSION

1. To conclude, bronchial artery embolization is a safe and simple procedure not associated with any significant complication.

2. The success rate of BAE is about 74% for the first six month follow up period as per our study.

3. BAE clearly reduces the mortality rate in hemoptysis and hence can be done for all cases provided the facility and trained personnel are available.

4. According to our study cavitatory lesions were found to be the common source of bleed in hemoptysis patients.

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STUDY PROFORMA

Name :

Age/Sex :

Address :

Annual Income :

Socio Economic Class :

MRD No. :

Study Id No. :

Clinical Data :

History :

Presenting Complaints :

Cough / Expectoration / chest pain / fever / loss of appetite / loss of weight / Hemoptysis / breathlessness / foul smelling sputum / blood stained sputum.

Past History :

Diabetes Mellitus / Systemic hypertension / Cardiac disease / PT / Bleeding Diathesis

Treatment history :

Personal history :

Smoking / Alcohol / menstrual complaints for female patients.

PHYSICAL EXAMINATION :

General examination / General well being / dyspnoea / pallor / jaundice / pedal edema / Lymphadenopathy / Clubbing.

Vital Signs :

Pulse rate : BP :

Respiratory rate : Temp :

JVP :

Systemic Examination :

CVS :

RS :

ABDOMEN :

LABORATORY DATA :

PRELIMINARY INVESTIGATIONS :

TEST	RESULT
Blood Sugar	
Blood Urea, Serum Creatinine	
CBC	
Serum Bilirubin	
Direct / indirect bilirubin	
AST/ALT	
SAP	
Total protein	
Serum albumin / globulin	
CXR PA View	
ECG	
USG Abdomen / CT	
Sputum AFB	

SPECIAL INVESTIGATIONS :

ECHO :

Sputum C/S :

CT / HRCT CHEST :

Bronchoscopy :

INFERENCE :

MASTER CHART

MASTER CHART- Hemoptysis causes

S. no	Age	Sex	LFT	RFT	CXR	Sputum for AFB	Sputum C/S	Sputum for malignant cells	ECHO	INR	CT/HRCT thorax	Broncho Scopy	Diagnosis
1	50	M	N	N	Ec Ch	neg	S.Pneu	-	-	-	Ec Ch	Ec Ch	B E
2	30	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
3	64	M	N	N	N	neg	NG	-	-	-	-	Br	Chr Br
4	55	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
5	53	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
6	52	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
7	47	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
8	26	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
9	64	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
10	49	M	N	N	Ca mg	-	-	-	MS with PHT	3.4	-	-	RHD
11	21	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
12	50	M	N	N	Con	neg	Kleb	-	-	-	Con	-	Pneum
13	62	M	N	N	N	neg	NG	-	-	-	-	Br	Chr br
14	30	M	N	N	Con	neg	Kleb	-	-	-	Con	-	Pneum
15	42	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
16	57	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
17	29	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
18	56	M	N	N	Ec Ch	neg	S.Pneu	-	-	-	Ec Ch	Ec Ch	B E
19	48	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
20	45	M	N	N	N	neg	NG	-	-	-	-	Br	Chr Br
21	60	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
22	38	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
23	54	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
24	70	M	N	N	Ho o	-	-	+	-	-	malig	GR	Ca Br
25	62	M	N	N	Infil+	+	neg	-	-	-	-	-	PT

S. no	Age	Sex	LFT	RFT	CXR	Sputum for AFB	Sputum C/S	Sputum for malignant cells	ECHO	INR	CT/HRCT thorax	Broncho Scopy	Diagnosis
26	22	M	N	N	Ca mg	-	-	-	MS with PHT	3.5	-	-	RHD
27	50	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
28	60	M	N	N	N	neg	NG	-	-	-	-	Br	Chr Br
29	30	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
30	57	M	N	N	Infil+	+	Neg	-	-	-	-	-	PT
31	70	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
32	45	M	N	N	Ec Ch	neg	S.Pneu	-	-	-	Ec Ch	Ec Ch	B E
33	30	M	N	N	Infil+	+	Neg	-	-	-	-	-	PT
34	40	M	N	N	Fb scar+	neg	Neg	-	-	-	-	-	PT seq
35	45	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
36	60	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
37	50	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
38	47	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
39	65	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
40	45	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
41	56	F	N	N	N	neg	NG	-	-	-	-	Br	Chr Br
42	47	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
43	23	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
44	45	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
45	35	F	N	N	LA	neg	Kleb	-	-	-	LA	-	L A
46	40	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
47	45	F	N	N	Ca mg	-	-	-	MS with PHT	3.9	-	-	RHD
48	39	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
49	55	F	N	N	N	neg	NG	-	-	-	-	Br	Chr br
50	42	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
51	70	F	N	N	Infil+	+	neg	-	-	-	-	-	PT

S. no	Age	Sex	LFT	RFT	CXR	Sputum for AFB	Sputum C/S	Sputum for malignant cells	ECHO	INR	CT/HRCT thorax	Broncho Scopy	Diagnosis
52	14	F	N	N	Ho o	-	-	+	-	-	malig	GR	Ca Br
53	45	F	N	N	Ho o	-	-	+	-	-	malig	GR	Ca Br
54	48	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
55	57	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
56	27	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
57	45	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
58	28	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
59	60	F	N	N	Cavity with crescent sign	neg	neg	-	-	-	Cavity with crescent sign	-	Asper
60	60	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
61	42	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
62	40	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
63	63	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
64	75	F	N	N	Ec Ch	neg	S.Pneu	-	-	-	Ec Ch	Ec Ch	B E
65	40	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
66	50	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
67	45	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
68	43	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
69	27	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
70	65	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
71	42	F	N	N	Fb scar+	Neg	neg	-	-	-	-	-	PT seq
72	24	F	N	N	Ca mg	-	-	-	MS with PHT	3.6	-	-	RHD
73	30	F	N	N	Infil+	+	Neg	-	-	-	-	-	PT
74	19	F	N	N	Infil+	+	Neg	-	-	-	-	-	PT
75	65	F	N	N	Infil+	+	Neg	-	-	-	-	-	PT
76	50	F	N	N	Infil+	+	neg	-	-	-	-	-	PT

S. no	Age	Sex	LFT	RFT	CXR	Sputum for AFB	Sputum C/S	Sputum for malignant cells	ECHO	INR	CT/HRCT thorax	Broncho Scopy	Diagnosis
77	27	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
78	50	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
79	53	F	N	N	Infil+	+	Neg	-	-	-	-	-	PT
80	52	F	N	N	Fb scar+	neg	Neg	-	-	-	-	-	PT seq
81	25	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
82	60	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
83	50	F	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
84	70	F	N	N	Ec Ch	neg	S.Pneu	-	-	-	Ec Ch	Ec Ch	B E
85	21	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
86	67	F	N	N	Ho o	-	-	+	-	-	malig	GR	Ca br
87	45	F	N	N	Infil+	+	neg	-	-	-	-	-	PT
88	61	M	N	N	Fb scar+	Neg	neg	-	-	-	-	-	PT seq
89	35	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
90	47	M	N	N	Ec Ch	Neg	Kleb	-	-	-	Ec Ch	Ec Ch	B E
91	52	M	N	N	N	Neg	NG	-	-	-	-	Br	Chr Br
92	37	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
93	50	M	N	N	Fb scar+	Neg	neg	-	-	-	-	-	PT seq
94	62	M	N	N	Ho o	-	-	+	-	-	malig	GR	Ca br
95	30	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
96	63	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq
97	42	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
98	32	M	N	N	Infil+	+	neg	-	-	-	-	-	PT
99	48	M	N	N	LA	neg	Kleb	-	-	-	LA	-	L A
100	30	M	N	N	Fb scar+	neg	neg	-	-	-	-	-	PT seq

Bronchial Artery Embolisation(BAE) – Follow up study (6 months)

S No	Age	Sex	BAE procedure	Post procedural hemoptysis	RFT	CT thorax
1	65	M	N	A	N	R UL cavity
2	50	F	N	Present	N	R UL cavity
3	57	F	N	A	N	R UL cavity
4	45	M	N	A	N	Non cavitatory
5	45	F	N	A	N	Non cavitatory
6	50	M	N	Present	N	R UL cavity
7	33	M	Failure	A	N	Non cavitatory
8	28	M	N	Present	N	Non cavitatory
9	43	M	N	A	N	R UL cavity
10	32	M	N	A	N	L UL cavity
11	50	M	N	A	N	L UL cavity
12	29	M	N	A	N	R UL, ML cavity
13	16	M	N	A	N	L UL cavity
14	40	M	N	A	N	Non cavitatory
15	28	F	N	A	N	Non cavitatory
16	43	M	N	Present	N	Non cavitatory
17	37	M	N	A	N	Non cavitatory
18	29	M	N	A	N	R UL cavity
19	27	F	N	A	N	R UL cavity
20	22	M	N	Present	N	L UL cavity

A - Absent *N - Normal*

ABBREVIATION

Infil+	- Infiltrates
Fb scar+	- Fibrotic scar
LA	- Lung Abscess
Con	- Consolidation
Ho o	- Homogenous opacity
Ec ch	- Ectatic changes
Ca mg	- Cardiomegaly
Neg	- Negative
Kleb	- Klebsiella
S. Pne	- Streptococcus pneumoniae
NG	- No Growth
Malig	- Malignancy
N	- Normal
Br	- Bronchitis
GR	- Growth
PT	- Pulmonary tuberculosis
RHD	- Rheumatic heart disease
Chr Br	- Chronic bronchitis
Ca Br	- Carcinoma Bronchus

PT seq	-	Pulmonary Tuberculosis sequelae
B E	-	Bronchiectasis
Pneum	-	Pneumonia
LA	-	Lung abscess
Asper	-	Aspergilloma
R	-	Right
L	-	Left
UL	-	Upper Lobe
ML	-	Middle Lobe
LL	-	Lower Lobe